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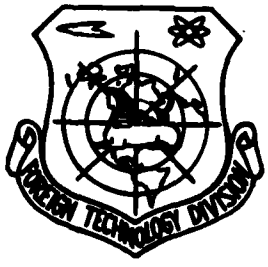
NEUROPSYCHIC DISORDERS IN TRICHLORFON POISONING

by

V. I. Barabash



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Block	Italic	Transliteration	Block	Italic	Transliteration
А а	*А а*	A, a	Р р	*Р р*	R, r
Б б	*Б б*	B, b	С с	*С с*	S, s
В в	*В в*	V, v	Т т	*Т т*	T, t
Г г	*Г г*	G, g	У у	*У у*	U, u
Д д	*Д д*	D, d	Ф ф	*Ф ф*	F, f
Е е	*Е е*	Ye, ye; E, e*	Х х	*Х х*	Kh, kh
Ж ж	*Ж ж*	Zh, zh	Ц ц	*Ц ц*	Ts, ts
З з	*З з*	Z, z	Ч ч	*Ч ч*	Ch, ch
И и	*И и*	I, i	Ш ш	*Ш ш*	Sh, sh
Й й	*Й й*	Y, y	Щ щ	*Щ щ*	Shch, shch
К к	*К к*	K, k	Ъ ъ	*Ъ ъ*	"
Л л	*Л л*	L, l	Ы ы	*Ы ы*	Y, y
М м	*М м*	M, m	Ь ь	*Ь ь*	'
Н н	*Н н*	N, n	Э э	*Э э*	E, e
О о	*О о*	O, o	Ю ю	*Ю ю*	Yu, yu
П п	*П п*	P, p	Я я	*Я я*	Ya, ya

*ye initially, after vowels, and after ъ, ь; e elsewhere.
When written as ё in Russian, transliterate as yё or ё.

RUSSIAN AND ENGLISH TRIGONOMETRIC FUNCTIONS

Russian	English	Russian	English	Russian	English
sin	sin	sh	sinh	arc sh	$\sinh^{-1}$
cos	cos	ch	cosh	arc ch	$\cosh^{-1}$
tg	tan	th	tanh	arc th	$\tanh^{-1}$
ctg	cot	cth	coth	arc cth	$\coth^{-1}$
sec	sec	sch	sech	arc sch	sech^{-1}
cosec	csc	csch	csch	arc csch	csch^{-1}

Russian	English
rot	curl
lg	log

NEUROPSYCHIC DISORDERS IN TRICHLORFON POISONING

V. I. Barabash, (Leningrad)

Summary. In the process of a clinical study of 82 patients hospitalized with trichlorfon poisoning it was established that in the acute period of intoxication neuropsychic disorders are expressed in syndromes characteristic of exogenous psychoses. With the disappearance of psychotic symptoms, pronounced asthenic conditions and a large number of neurotic complaints were observed, especially in severe and moderate cases of poisoning. In a number of cases there were symptoms of polyneuritis with motor and sensory disorders, while mental changes were expressed in persistent intellectual decline and memory impairment of a psycho-organic type. In profound intoxications the mental disorders usually speak in favor of a developing toxic encephalopathy.

Trichlorfon, an effective insecticide, is widely used in the nation's economy and homes. Being a highly toxic substance of the organophosphorous series, trichlorfon can cause acute and chronic poisoning if not used properly.

The chemical features of organophosphorous insecticides and their effects on animals and man have been described in some detail [1-4]. There have been reports about clinical cases of acute poisoning and death caused by these substances. Chronic symptoms have also been described [5-12]. Neurologic disorders occurring in patients with

acute trichlorfon poisoning have been rather thoroughly studied and described by V. S. Lobzin and P. Ye. Tsinovoy [13]. A. A. Askarov has observed chronic poisoning by the organophosphorous insecticide demeton [14]. It is seen from the descriptions given by him that in a number of cases the symptoms of acute intoxication give way to persistent asthenic symptoms of an organic character with pronounced mental disturbances.

We studied the effect of trichlorfon intoxication on the neuropsychic condition in 82 patients ranging in age from 23 to 52, who were admitted for hospital care after accidental or suicidal poisoning with this substance. The patients had drunk 50 to 200 grams of 30% solution of trichlorfon. A developed pattern of mental disorders was observed in 48 patients.

The acute period of poisoning was expressed in general weakness, dizziness, headache, miosis, pallor, especially in the face, scleral hyperemia, nausea and frequent vomiting, and severe pains in the stomach; sometimes observed were diarrhea, absence of pupillary response to light, painfulness of the eyeballs during convergence, and other symptoms of acute intoxication.

Neuropsychic disturbances were noted in all persons who had taken trichlorfon. The nature of these disturbances varied, however, with the dose received, the amount of time elapsed before help was given, and other factors (when food was last taken before the poisoning, how soon vomiting began, and the patient's general condition.

Forty-five of the patients had neurosis-like disorders, which, forming against a background of symptoms of general intoxication, included neurotic behavior associated with the very fact of poisoning. The primary (neurotic) reaction also reflected the victim's personality. Patients were either listless, sullen, and withdrawn, or were agitated, alarmed, fidgety, demanded help, and blamed themselves. In some cases mild euphoric states were noted. This reaction lasted from a few dozen minutes to 1-2 hours. In cases of massive intoxication more severe disorders developed in the patients.

Twenty-eight patients were psychically stunned. They were characterized by general sluggishness, indifference to persons and activ-

ities around them, including medical measures; they submitted passively to the actions of medical personnel; left to themselves they remained in bed for long periods without changing position; their facial expression was sleepy; they gave one-word answers to questions asked persistently and in a loud voice. They did not demonstrate a deep understanding of their situation, nor did they engage actively in conversation. Their movements were sluggish and slow, their actions automatic. As their general condition deteriorated and intoxication increased, 15 patients exhibited confusion and motor excitation. The changed state of awareness was accompanied by alarm and fear, as well as illusory and elementary hallucinatory disturbances. Six patients had sporadic delusions, usually delusions of action or persecution. The behavior of the patients reflected characteristics of psychotic disturbances. In 4 cases symptoms of disturbed consciousness were combined with mania. Only superficially oriented in their surroundings, the patients did not recognize persons familiar to them and did not clearly perceive events taking place in their presence. Their speech was rapid, their judgement inconsistent. Correct answers alternated with irrelevant statements. Foolish behavior and speech were noted.

In addition to being psychically stunned and confused, 2 patients developed epileptiform disorders: slight convulsive movements of the hands and head accompanied by mild tonicity of the body. The intensity of the spasms was less pronounced then that observed in classic epilepsy; other disturbances characteristic of fully-developed seizures (biting of the tongue, facial cyanosis, etc.) were not observed.

Extremely severe poisoning was characterized by the syndromes of loss of psychic activity: a soporous or comatose state.

Ten patients with mental disturbances had delirium or amential delirium syndromes. It should be noted that 6 persons with delirious psychosis suffered from alcoholism or systematic alcoholic abuse.

Development of the delirium syndrome was preceded by dysphoria, alarm, and fear. Along with a partial loss of orientation in their environment the patients experienced true visual hallucinations, often related to their work or home, but there were also cases of

visual zoohallucinations. Auditory hallucinations were fragmentary (the sound of "Morse code", the noise of running machines, etc.). The statements of patients suggested specific delusions of action and persecution. Signs of mentism were also observed. These patients were extremely restless. All of them were transferred to a psychiatric ward after certain imperative measures had been taken.

The delirious states lasted 5-7 days and gave way to a set of pronounced asthenic symptoms; some of the patients displayed polyneuritic disorders.

Patients with amential delirium were characterized by more pronounced confusion and motor and speech excitation, as well as disorientation. The general condition of the patients was serious (dehydration, drawn features, dryness in the mouth, febrile body temperature, etc.). The amential disturbances lasted 5 to 10 days, after which severe asthenia was detected. One patient developed distinct amnestic disturbances of the type found in Korsakov's syndrome, along with paresthesia in the distal portions of the upper and lower extremities.

The cases of trichlorfon poisoning which we studied were serious and were accompanied as a rule by a more or less pronounced asthenic condition. Asthenic disorders were sometimes manifested in loss of inhibitions and euphoria. Patients were moderately excited, difficult for medical personnel, underestimated the seriousness of their situation, tried to obtain early release, and so forth. After some time the euphoric stage gave way to symptoms typical of asthenia. Patients became listless and did not move around, showed little interest in reading or associating with others, spent a great deal of time in bed, and easily became tired during conversation. There were cases where asthenic disorders were combined with depressive, obsessive, and hypochondriac symptoms. The hypochondria was usually expressed in the form of complaints about deteriorating eyesight, difficulty in breathing, heart trouble, and unusual weakness which did not correspond to the actual somatic condition of the patient.

In severe and moderate poisoning the psychotic disturbances were replaced by very pronounced asthenia with more or less distinct signs of weakened memory (with respect to current events, work habits,

and literature). Patients in this group also exhibited transient polyneuritic disorders, but 5 patients, including some who had experienced delirium, showed marked symptoms of toxic polyneuritis. Patients with severe polyneuritis, extreme pain, or motor disturbances were transferred to the nervous disease clinic for special treatment. The polyneuritic symptoms in the patients observed by us were usually found 15-20 days after the poisoning. At first they were exhibited as unpleasant sensations in the distal portions of the extremities and as periodic pain and weakness in the arms and legs. Gradually the pains became intense, and there was definite pain along the path of nerve trunks. In some cases the pains were excruciating. Patients developed flaccid paresis and paralysis of the muscles in the upper and lower extremities, along with a reduction in all types of surface sensitivity. Acute polyneuritic symptoms lasted $1\frac{1}{2}$-2 months, while residual effects were noted in the distal portions of the lower extremities for a year or more.

The asthenic disturbances developing after trichlorfon poisoning were combined with pronounced vegeto-vascular disorders (acrocyanosis, unstable arterial pressure, vasomotor play, tachycardia).

The symptoms of asthenia were characterized by regressiveness. The patients fully recovered in 20-40 days. It should be noted that in 6 persons the asthenic syndrome developed after the state of health had improved, i.e., after 2-4 months or more, and was characterized by persistence, a progressive wavelike course, and symptoms of organic brain lesions. Along with asthenic symptoms these patients exhibited a diminution of intellectual powers and volition, forgetfulness, absent-mindedness, unusual tendency to fatigue, and a constant feeling of "inner tension". Persistent interruptions of sleep and frequency of nightmares also figured prominently in the complaints of patients. Emotional distress was also more salient in this group of patients: increased irritability, unstable moods, sensitivity. The patients had a low tolerance for noise and strong smells, unusual reactions to certain drugs, and increased susceptibility to alcohol. Two patients developed uncharacteristic timidity and obsessive fears. Attention was drawn to the persistence of autonomic disorders in these patients. Complaints of impotence were not infrequent.

Depressive feelings were also experienced (inferiority, reticence, an urge to seek seclusion).

The decompensation period was characterized by hypochondriac complaints, especially in patients with sensory and motor disorders and symptoms of paresthesia.

We traced 3 cases of the formation of a psychoorganic syndrome after severe trichlorfon intoxication. In all 3 patients the late-period asthenic disturbances which were mentioned above preceded organic symptoms. Predominant in the asthenia were increased irritability, tendency to anger, and affective reactions. The patients themselves noted these changes in their nature. Mild dysphoric conditions were subsequently revealed: unstable moods, anxiety, tension, irritability, tendency to cry. This made it difficult for the patients to do their work. They were emotionally drained, their attention became fixed, and they were less able to do creative (intellectual) work and assimilate new material. Later on more pronounced changes were observed: the use of stereotyped answers and phrases, and a tendency to burden one's speech with details. In time these persons were forced to take simpler work. Their interests were limited to personal cares and worries about health.

Neuropsychic disorders were especially persistent when they developed in persons with encephalopathy (as a result of severe poisoning).

Under unfavorable living conditions or an excessive load (prolonged neuropsychic stress, physical exhaustion, infections, alcoholic intoxication) satisfactory compensation was disrupted, and marked neurotic or even psychotic syndromes appeared. With rational specialized employment and periodic treatment these patients retained the ability to work, even if at a lower professional level.

Our observations are therefore evidence of the pronounced neurotropic and psychotropic effect of organophosphorous insecticides. The acute period of trichlorfon poisoning is expressed in syndromes of acute exogenous psychosis. Psychic disturbances characteristic of toxic encephalopathy are later observed in cases of serious intoxication. Our data confirm the opinion in the literature that the organophosphorous insecticides belong to the group of neurodestructive poisons [12].

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